dependent manner. AT1R silence effectively blocked Ang II-induced damage. MitoTEMPO attenuated the activation of NRLP3 inflammasome through clearance of reactive oxygen species (ROS). Moreover, Ang II-induced mitochondrial dysfunction was markedly inhibited by silence of NLRP3.
Conclusion: Ang II stimulation induces NLRP3 inflammasome activation through AT1a receptor. Ang II-induced NLRP3 activation is mediated by mitochondrial dysfunction, with overproduction and accumulation of ROS. NLRP3 inflammasome activation plays an important role in kidney injury, and blocking it can be a potential therapeutic target for hypertension-associated kidney damage.

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0220
International Congress of Chinese Nephrologists: Clinical and Pathological Characteristics of 75 Chinese Patients with Benign Hypertensive Nephrosclerosis

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Objective: To Identify the clinical and pathological characteristics of 75 Chinese patients with benign hypertensive nephrosclerosis (BHN) and the relationship between the clinical and pathological changes.
Methods: BHN patients proved by renal biopsy were retrospectively analyzed based on the clinical and pathological records. Glomerular lesions were divided into five types: hypertrophic, focal segmental sclerotic, solidified or ischemic (including wrinkled and obsolescent changes). Patients were grouped for further analysis according to whether eGFR $\geq$ 60 ml/min/1.73 m^2, $\geq$ 30% ischemic glomeruli, $\geq$ 20% obsolescent glomeruli, respectively.
Results: 75 BHN patients with an average age of 44.4 $\pm$ 10.7 years, M/F 4:1. eGFR had significant negative correlation with systolic blood pressure (SBP) ($r = -0.276$, $P = 0.016$) but not diastolic blood pressure (DBP). DBP had significant negative correlation with age ($r = -0.3$, $P = 0.007$). The most common pathological type of glomerular lesion was ischemic (30.6%), including ischemic wrinkled (13%) and obsolescent (17.6%). Arteriolar stenosis had significant positive correlation with ischemic glomerular lesion ($r = 0.33$, $P = 0.004$), but not obsolescent glomeruli. Multivariate analysis showed: (1) SBP rather than DBP was independent risk factor of eGFR decline; (2) independent associated factors of ischemic glomerular change were TG (OR = 1.78, 95% CI 1.02~3.1; P = 0.041), age (OR = 0.93, 95% CI 0.89~0.99; P = 0.018) and eGFR (OR = 0.97, 95% CI 0.95~0.99; P = 0.004); (3) independent associated factors of obsolescent glomeruli were TG (OR = 1.96, 95% CI 1.13~3.4; P = 0.016) and eGFR (OR = 0.97, 95% CI 0.95~0.99; P = 0.012).
Conclusion: SBP is independent risk factor of eGFR decline in BHN. TG promoted the development of ischemic and obsolescent glomerular lesion which may offer a new therapeutic target for the management of BHN.

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0235
Risk Factors and Their Interaction on CKD in Patients with Hypertension and DM: A Multicenter Case Control Study in Taiwan

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Objective: Chronic kidney disease (CKD) is highly prevalent in Taiwan. More than two-thirds of end-stage renal disease is associated with diabetes mellitus (DM) or hypertension (HTN). Therefore, the formulation of a special preventative policy of CKD in these patients is essential. This study surveyed 14 traditional risk factors and identified their effects on CKD in patients with HTN/DM and compared these with their effects in the general population.
Methods: This study included 5328 cases and 5135 controls in the CKD/HTN/DM outpatient centers of 10 hospitals in Taiwan. Fourteen common effect factors were surveyed (four demographic, five disease and five lifestyle), and their effects on CKD were tested. Significance tests were adjusted by the Bonferroni method. Results of the stratified analyses in the variables were presented with significant heterogeneity between patients with different comorbidities.
Results: Male, ageing, low income, hyperuricemia and lack of exercise habits were risk factors for CKD, and their effects in people with different comorbidities were identical. Anemia was a risk factor, and there was an additive effect between anemia and HTN on CKD. Patients with anaemia had a higher risk when associated with HTN [odds ratio (OR) = 6.75, 95% CI 4.76—9.68] but had a smaller effect in people without HTN (OR 2.83, 95% CI 2.16—3.67). The association between hyperlipidaemia-related factors and CKD was also moderated by HTN. It was a significant risk factor in people without HTN (OR = 1.67, 95% CI 1.38—2.01) but not in patients with HTN (OR = 1.03, 95% CI 0.89—1.19). Hepatitis B, hepatitis C, betel nut chewing, smoking, alcohol intake and groundwater use were not associated with CKD in multivariate analysis.
Conclusion: We considered that patients with HTN and anemia were a high CKD risk population. Physicians with anemic patients in outpatient clinics need to recognize that patients who have HTN might be latent CKD cases.

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0240
Arterial Stiffness Evaluated by Carotid-Femoral Pulse Wave Velocity Increases the Risk of Chronic Kidney Disease in a Chinese Population-based Cohort

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Objective: Chronic kidney disease (CKD) is an independent risk factor for cardiovascular disease. Although pulse wave velocity (PWV), which reflects arterial stiffness, was increased in subjects with CKD, little is known regarding whether arterial stiffness can increase the risk of CKD. To help clarify this, we conducted a prospective cohort study to measure the association of arterial stiffness with CKD.
Methods: A total of 7455 adults who visited the Health Checkup Clinic consecutively were enrolled from April 2010 to December 2010. 92 participants with proteinuria, and 108 participants with decreased eGFR calculated by the CKD-EPI equation (eGFR $<$ 60 ml/min/1.73 m^2) at baseline were excluded. CKD was defined as decreased eGFR or presence of proteinuria (urine protein $\geq$ 1+). During the follow-up, 43 participants were lost to follow-up. 58 participants were excluded from ananlysis due to insufficient blood or urine samples, 7154 participants aged 54.8 $\pm$ 10.6 years without CKD at baseline were enrolled in the final ananlysis. Arterial stiffness was evaluated by carotid-femoral PWV (cfPWV) using the SphygmoCor device (AtCor Medical Ltd., Sydney, Australia).
Results: During 3 years of follow-up, 167 (2.3%) patients developed CKD, 101 (1.4%) patients with proteinuria and 68 (1.0%) patients with decreased eGFR. After adjusting for potential confounders, either cfPWV (per 1cm/s increase) or the highest quartile of cfPWV (increased cfPWV) was independently associated with increased risk of proteinuria and CKD ($P < 0.05$). By contrast, neither cfPWV (per 1 cm/s increase) nor increased cfPWV was associatied with decreased eGFR in the multivariable logistic regression analysis.
Conclusion: This study revealed that arterial stiffness increases the risk of CKD. Hence, high-risk patients with arterial stiffness should have targeted monitoring for the development of CKD.